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# Foreign Animal Disease Report

United States  
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### **Emergency Programs Activities**

**Field Investigations.** During the first quarter of Fiscal Year (FY) 1991 (October 1, 1990, to December 31, 1990), veterinarians from the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), Veterinary Services (VS) and from State departments of agriculture conducted 48 investigations of suspected foreign animal diseases in the United States and Puerto Rico to eliminate the possibility that an exotic disease may have been introduced into the United States. These investigations included 39 for vesicular conditions, 1 for swine septicemic conditions, 1 for mucosal conditions, 1 for exotic Newcastle disease in poultry, 1 for encephalitic conditions, and 5 for screwworms or undesignated conditions. No foreign animal diseases or pests were found.

**Training.** During November 5–9, 1990, 19 teachers of infectious diseases at veterinary colleges and diagnosticians at State laboratories received training at Plum Island, NY, to increase their awareness of foreign animal diseases and provide the teachers with procedures for reporting suspicious cases of foreign animal diseases to State and Federal officials.

Foreign animal disease diagnosticians in the VS Western and Central Regions attended seminars designed to increase their knowledge and skills in the recognition and diagnosis of foreign animal diseases. These seminars were held January 15–17 at Salt Lake City, UT, and February 5–7 at Tulsa, OK.

**Educational Exhibit for International Travelers.** In October 1990, APHIS presented to the World Dairy Expo in Madison, WI, the agency's new educational exhibit on the risks of animal disease transmission by international travelers. APHIS is now preparing a video that can be used with the exhibit in future presentations. The video should be completed by September 1991.

**Biosecurity Exhibit and Videos.** The APHIS educational exhibit and eight videos on poultry biosecurity mentioned in the Spring 1989 issue were shown at the 1991 Southeastern Poultry and Egg Association Show in Atlanta and the Midwest Poultry Association Show in Minneapolis.

The videos are entitled  
What is Biosecurity?  
Broiler Operations: All In—All Out  
Egg-Laying Operations: the Commercial Farm  
Live Poultry Markets: the Crossroads  
Hatcheries: Babies Are Our Business  
Transportation and Feed Mills: Transfer Points for Disease  
Turkey Operations: Year Round Production  
Game Birds and Small Flocks

Arrangements for viewing the videos can be made by contacting the VS Area office in your State, or your nearest USDA Extension Service office. Copies of the videos may also be purchased from the National Audiovisual Center, 8700 Edgeworth Drive, Capitol Heights, MD 20743-3701. The center is equipped to handle orders via FAX (301-763-6025) or voice contact (800-638-1300).

**Bovine Spongiform Encephalopathy (BSE) Surveillance.** Since 1981, 459 head of cattle (101 males and 358 females) have been imported into the United States from Great Britain and Ireland. (A total of 462 head was reported in the Winter 1990 issue. Three of these cattle did not actually enter the United States.) All States have reported their initial BSE surveillance activities. By February 1991, 214 head (63 males and 151 females) had been examined and found negative for any evidence of BSE, and 69 head (37 males and 32 females) had been slaughtered or killed with no indication of central nervous system (CNS) abnormalities. A bull from the United Kingdom was slaughtered in 1988 because of clinical signs that indicated CNS abnormalities, but necropsy revealed no evidence of BSE. Traceback activities continue, including efforts to locate 175 cattle that have been sold to new owners in different States.

(Dr. M.A. Mixson, Emergency Programs, VS, APHIS, USDA, Hyattsville, MD 20782, 301-436-8073)

**Exotic Newcastle Disease in Water Birds** Deaths among double-crested cormorants (*Phalacrocorax auritus*) and white pelicans (*Pelecanus erythrorhynchos*) due to exotic viscerotropic velogenic Newcastle disease were diagnosed in northern Saskatchewan, Canada, in August and September 1990. Losses also were encountered in cormorant populations in the Provinces of Alberta and Manitoba, but no virus was isolated. Mortality estimates included approximately 6,000 cormorants and 100 white pelicans. An undetermined number of ring-billed gulls (*Larus delawarensis*) also were involved.

Most of the cormorant losses were in full-sized young-of-the-year. A predominant clinical sign described in these birds was paralysis of one wing, with the wing held against the body. Less commonly, birds had paralysis of a leg or held their head in an unusual position. Because velogenic Newcastle disease is particularly dangerous to domestic poultry, anyone who encounters or handles sick cormorants, white pelicans, or gulls should avoid cross-contamination to chickens or turkeys.

The species of birds that were exposed to the virus on the breeding grounds in northern Saskatchewan move into the United States or farther south to winter. Wildlife authorities have been alerted concerning the disease outbreak. They have been encouraged to submit sick or dead birds promptly to a State or Federal diagnostic laboratory for appropriate testing. Foreign animal disease diagnosticians should be prepared to assist wildlife biologists with investigation of illness in these birds.

(Dr. Victor F. Nettles, Southeastern Cooperative Wildlife Disease Study, University of Georgia, Athens, GA 30602, 404-542-1741)

**Equine Influenza in China** A new influenza A virus (subtype H3N8) in China caused severe respiratory disease and death in horses during 1989 and severe respiratory disease in 1990. In March 1989, an outbreak of respiratory disease occurred in horses in the Jilin and Heilongjiang Provinces of northeastern China (Guo, Y., et al. 1990. Chinese J. Exper. Clin. Virol. 3: 318–322), causing 81 percent morbidity and up to 20 percent mortality in some affected herds. In Zhilia County, Jilin Province, mortality reached 35 percent among 13,151 infected horses. The outbreak peaked in late March and April 1989 and abated by June. Onset of the disease was acute, with coughing, bronchitis, pneumonia, and fever of more than 40 °C. Affected animals were fatigued and had inflamed nasal mucosa and copious mucous discharge from the eyes and nose. In most cases, the course lasted 5 to 6 days, but some horses showed signs for 2 weeks. Death was associated with pneumonia and enteritis. Similar signs of disease were noted in mules but not in donkeys. None of the people who worked with the diseased horses had signs or symptoms of respiratory disease. The disease reappeared during April 1990 in Heilongjiang Province, resulting in 41 percent morbidity but no mortality.

An influenza virus of the H3N8 subtype was isolated from the infected horses and was shown to be antigenically and molecularly distant from the equine 2 (H3N8) viruses currently circulating in the world. The available evidence suggests that the new equine influenza virus in northeastern China is the latest mammalian influenza virus to emerge from the avian influenza gene pool in nature and that it spread to horses without genetic reassortment. (See 14-2: 6-7.)

Will the outbreak in horses be limited to China or become global? The answer will depend upon the movement of horses from that part of the world. Because there is trade across the Sino-Soviet border, the most likely route for spread is through the Mongolian horse population to central Russia.

The world horse community should be aware of the presence of this virus and maintain quarantine practices to reduce its spread. One important consideration is that, although the virus originated recently from avian species, it seems to have lost its ability to replicate in ducks, thereby reducing the likelihood of spread by aquatic birds during migratory flights.

(Robert G. Webster and Yoshihiro Kawaoka, Department of Virology and Molecular Biology, St. Jude Children's Hospital, 332 N. Lauderdale, P.O. Box 318, Memphis, TN 38101; and Guo Yuanji, National Influenza Centre, Department of Influenza, Institute of Virology, Chinese Academy of Preventive Medicine, Beijing 100052, China)

## Foreign Animal Disease Update

This update article is a summary of disease surveillance information taken from various sources, including bulletins of the Office International des Epizooties (OIE), covering July, August, and September 1990.

There were reports of 1,902 occurrences of **foot-and-mouth disease** (FMD) in the OIE regions of South America (1,215), Africa (282), Europe (222), and Asia (183). Among the FMD viruses isolated, 36 percent were type O; 5 percent were type A; 0.3 percent, C; 0.1 percent, SAT 1; and 0.2 percent, Asia 1. Approximately 58 percent were untyped.

In Africa, Algeria reported 223 outbreaks of FMD type O for June and 53 for July. Samples submitted by Ethiopia to the World Reference Laboratory for Foot-and-Mouth Disease, Pirbright, UK, for September contained type O and SAT 1 activity. The director of Moroccan Animal Husbandry and Veterinary Services reported an outbreak of FMD in sheep along the Algerian border. The initial occurrence, on December 21, was characterized by lameness in adult animals and sudden death in very young lambs, caused by an FMD virus that the Pirbright laboratory identified as type O. The outbreak was located in a buffer zone where preventive vaccination of sheep (monovalent Tunisian type O 229) and cattle (bivalent type A5–Tunisian O 229) has been ongoing since December 11, 1990. The vaccination program followed the identification of FMD in the Wilayas area of western Algeria bordering four east Moroccan provinces. Farm animals cannot be moved within and from the buffer zone. FMD type O virus was last isolated in Morocco in 1965. That country's most recent prior outbreak of FMD in sheep involved type A virus in 1983. Chad and Burkina reported untyped FMD outbreaks for May through July and August, respectively. On November 2, 1990, Togo reported three outbreaks of FMD involving over 1,000 cases in the Golfe and Yoto prefectures. Togolese animal health services suspect that the causal agent was introduced by livestock imported from the Sahel and spread with commercial livestock. Neighboring Ghana also reported three suspected outbreaks of FMD. Specimens were submitted to the Pirbright laboratory for typing. Cote-d'Ivoire, which shares a border with Ghana, reported four outbreaks of FMD.

In South America, Argentina reported 667 outbreaks of FMD for January through July 1990, with 115 type O, 34 type A, 1 type C, and 517 untyped. Colombia reported 13 outbreaks of type O FMD and 50 of type A for June, July, and August. Venezuela reported an outbreak of FMD type A in April 1990. Paraguay and Uruguay each reported an outbreak of untyped FMD in September. Brazil reported 469 outbreaks of untyped FMD for March through August and occurrences of types O (5), A (5), and C (2) without indicating outbreaks.

In Asia (the Middle East, Asia proper, and the Far East), Iran reported 44 outbreaks of untyped FMD for the first 3 months of 1990 and sporadic occurrences of types O and A. Oman had 107 outbreaks of FMD—41 outbreaks of type O for July and August and 66 untyped outbreaks in the same period. The Pirbright laboratory identified type O from Saudi Arabia and SAT 1 from Yemen. FMD types O, A, C, and Asia 1 were recorded in Pakistan during July and August. FMD type A was identified from Nepal in July; type O was identified from Bhutan in August and from Nepal in September. Four outbreaks of untyped FMD and the presence of disease due to types O, A, and Asia 1 were reported from Myanmar in June and July. Hong Kong experienced two outbreaks of untyped FMD in June. Kampuchea reported one occurrence of FMD type Asia 1. Turkey reported 218 outbreaks of type O FMD from June through August and 3 outbreaks of type A during June and July.

The U.S.S.R. reported an outbreak of FMD type A in March 1990.

Mexico reported five outbreaks of **vesicular stomatitis** (VS) for June and August.

In Central America, Guatemala, Belize, Honduras, El Salvador, and Costa Rica reported a total of 36 outbreaks of VS serotype New Jersey (VS–NJ) during spring and early summer 1990. El Salvador reported Indiana serotype (VS–IN) in May. VS–IN predominated in Panama, with four occurrences in July and August. Two outbreaks of VS–NJ were also reported there, one in June and one in July.

In June through August, in South America, Colombia reported 50 outbreaks of VS-NJ and 35 of VS-IN. Venezuela reported VS-NJ outbreaks in March and May.

Sri Lanka reported 17 outbreaks of **rinderpest** (RP) for March through May 1990, killing 46 of the 260 cattle affected. Veterinary Services, Nairobi, Kenya, reported 100 cases of RP in the Rift Valley Province, involving approximately 10,000 animals, 20 of which died. Diagnosis was by the recognition of clinical signs and results of the agar gel immunodiffusion test.

Oman reported 31 outbreaks of **peste des petits ruminants** (PPR) for May through August. Numbers reported peaked in July, with 413 goats affected. Cote- d'Ivoire reported PPR activity for July and August.

Portugal reported 130 outbreaks of **contagious bovine pleuropneumonia** (CBP): 364 cattle were affected in May and 223, in June. Spain reported outbreaks in the Provinces of Guipuzcoa, Madrid, and Vizcaya. Six of the cases were confirmed by complement-fixation tests of specimens from a herd of 37 dairy cattle. The source of the infection is unknown.

Italy reported one comfirmed outbreak of CBP and a suspected outbreak from Bergamo Province. Results of a November 5 laboratory test confirmed the first outbreak of CBP in Italy since the disease was eradicated there in 1899. The number of possible cases in the suspected outbreak has not yet been determined, but the affected farm has 427 beef cattle. Control measures include slaughter and destruction of positive, suspected, and exposed animals, quarantine of premises, and delineation of infected and protection zones.

In Africa, Kenya reported six outbreaks of CBP for April, May, and June 1990, and Tanzania identified an outbreak in Ngorongoro during June. CBP was also reported from Cote-d'Ivoire, Kuwait, and Myanmar.

Madagascar reported that 35 cattle died in 37 outbreaks of **lumpy skin disease** (LSD) during March and April 1990. Zimbabwe reported 12 outbreaks of LSD during July through September involving 120 cattle. Kenya reported an outbreak of LSD in April, and Zambia reported an outbreak in February. Cote- d'Ivoire and South Africa reported LSD during the summer of 1990.

An outbreak of **Rift Valley fever** in Zimbabwe during August affecting six animals was the first reported from that country since June 1989.

**Bluetongue** was reported from South Africa and the United States through the summer months of 1990.

Turkey reported 201 outbreaks of **sheep and goat pox** (SGP) during June through August, in which 274 of 700,431 affected sheep and 138 of 4,979 affected goats died. Reports from Iran identified 83 SGP outbreaks during the first 3 months of 1990. Algeria reported 26 outbreaks from April through July, and Morocco reported 2 outbreaks affecting 520 sheep during June. Oman reported 15 outbreaks of SGP for May through August affecting 61 animals. Three March outbreaks reported by Kuwait involved 195 sheep. The disease was identified in the Cote-d'Ivoire during June and in Myanmar during August.

Spain and Morocco continued to report outbreaks of **African horse sickness** (AHS). (See 18-4: 5.) A November 5, 1990, report from Spain's Ministry of Agriculture, Fisheries and Food identified 56 AHS outbreaks in the Province of Malaga. A mid-November report from Morocco identified 51 outbreaks above the 34th parallel. Control measures in both countries included vaccination and restriction of animal movements. Namibia reported an AHS outbreak in July, and Zimbabwe reported outbreaks in August and September. South Africa acknowledged the presence of AHS in June and July.

A total of 150 outbreaks of **African swine fever** (ASF) were reported. Spain had 140 outbreaks of ASF in July and August affecting 16,269 swine. Italy had nine outbreaks: seven during June and August at Nuoro, one during July at Oristano, and one during September at Cagliari. Mozambique had an outbreak in September affecting 423 swine: 202 died, 119 were destroyed, and 102 were slaughtered for food.

European countries reporting **hog cholera** (HC) to OIE for the third quarter of 1990 included Austria, Belgium, Germany, Hungary, Italy, and Yugoslavia. After being free of the disease since May 1988, Czechoslovakia had 16 outbreaks of HC in August attributed to the movement of wild boars. The French Ministry of Agriculture and Forestry reported HC on the island of Corsica involving four animals. Two died, and two were destroyed. The U.S.S.R. reported six outbreaks of HC during April through June. In the Americas, Brazil, Mexico, Paraguay, and Chile reported 38, 24, 3, and 2 outbreaks, respectively. Argentina also had HC during June and July. Madagascar reported an outbreak of HC in March. In Asia, Taiwan, South Korea, the Philippines, and Hong Kong reported 11, 7, 6, and 5 outbreaks, respectively. Myanmar reported the presence of HC but no outbreaks for July and August.

Madagascar reported three **Teschen disease** (TD) outbreaks in March and two in April 1990, resulting in the death of 50 swine. The U.S.S.R. reported TD in April and June. The June outbreak resulted in the slaughter of 138 swine in the Ukraine.

Myanmar reported **fowl plague** for July and August.

In Africa, 26 **Newcastle disease** (ND) outbreaks were reported from Algeria, Egypt, Kenya, Madagascar, Namibia, and Zambia. (The virus was untyped and was assumed to be velogenic.) Cote-d'Ivoire noted disease activity but no outbreaks in July and August. In Europe, 4 outbreaks of ND were reported from Albania, 3 from the U.S.S.R., and 11 from Yugoslavia. An outbreak at Krasnodar, U.S.S.R., during May resulted in the destruction of 20,200 birds. There were seven outbreaks of ND in Turkey during July-August. ND outbreaks in Haiti during February and March 1990 killed 67,000 of 89,000 affected birds. ND outbreaks were reported from Brazil (76) and Mexico (4). Asian outbreaks of ND were reported from Iran, Kuwait, Myanmar, Hong Kong, and the Philippines. The disease was also reported from Myanmar, but no outbreaks were noted from there.

Three outbreaks of **velogenic viscerotropic Newcastle disease** (VVND) were reported in 1990: 1 from Botswana during July and August; 10 from peninsular Malaysia during January, March, April, and June, killing 23,159 of 43,230 affected birds; and 4 from South Korea during June, 2 during July, and 1 during August.

No new outbreaks of **viral hemorrhagic disease** (VHD) of rabbits have been reported in Mexico (see 18-4: 6-7) since the last cases were recorded there, October 10, 1990. Surveillance continues in that country, and apparently normal seropositive rabbits have been identified.

The Federal Veterinary Office in Berne, Switzerland, has notified the OIE that they confirmed **bovine spongiform encephalopathy** (BSE) November 3, 1990, in the Berne canton in a 6-year-old cow showing ataxia and hypersensitivity. Meat-meal of unknown origin is the suspected source of disease in this case. As a means to prevent additional cases, the Swiss Government is considering a ban on the feeding of meat-meal to ruminants. The Government has also included BSE on the list of notifiable diseases and required the incineration of carcasses of diseased animals.

On February 28, 1991, BSE was confirmed in a cow that died January 16, 1991, at Plouha, France, after showing signs of the disease. The herd of 68 dairy cows from which the affected cow came was purchased by the French National Center for Veterinary Pathology Studies for research to be carried out at Lyon.

The United Kingdom, Ireland, Oman, Switzerland, and France are the only countries known to have had cases of BSE. (See 18-4: 6, 18-3: 5, 18-2: 6, and 16-4: 4 ff.)

On February 25, 1991, the U.S.-Mexico Joint Commission for the Eradication of Screwworms declared the **screwworm** eradicated from Mexico. Attending the declaration ceremony were then-Secretary of Agriculture Clayton Yeutter and Mexican Minister of Agriculture Carlos Gonzalez. The present screwworm program continues to operate in Belize and Guatemala. Negotiations are underway for new cooperative agreements for the dispersal of sterile flies in El Salvador and Honduras.

(Dr. Peter Fernandez, International Services, APHIS, USDA, Hyattsville, MD 20782, 301-436-8892)

#### **Peace Corps Veterinarians in Morocco**

Five veterinarians are working in eastern and southern Morocco on 1-year assignments to the Volunteer Partner Program of the U.S. Peace Corps. This program is designed for professionals unable to commit themselves for a longer period, to encourage them to profit from the cultural, professional, and personal rewards of Peace Corps service.

Cooperative efforts among the Peace Corps, the American Veterinary Medical Association, and the Moroccan Government led to recruitment of the first group of nine veterinarians. They arrived in Rabat in August 1989 and received 7 weeks of training in Arabic and aspects of animal agriculture and health in Morocco. A second group of veterinarians is now pursuing its work, and the program is projected to run for another 3 years.

All participants are considered employees of the Moroccan Government and work in local livestock services in neglected arid and semiarid parts of the country. The professional activities of the 13 participants to date include working with local technicians and veterinarians to implement Government animal health programs, including vaccination campaigns, parasite treatments, meat inspection, and disease surveillance investigations; working with local dairy cooperatives to establish record systems, reproductive herd health programs, and mastitis prevention programs and to address other health concerns; and extension work with farmers, developing educational materials on sheep husbandry, mastitis prevention, and echinococcosis (a zoonotic disease with a high prevalence in the human population of Morocco).

Small Project Assistance grants are administered from the U.S. State Department's Agency for International Development to build feeders for a women's sheep cooperative in Morocco, buy tools for a foot care program, and implement nutrition programs in dairy cooperatives.

Animal diseases encountered in Morocco commonly reflect the underlying problem of inadequate nutrition, which may stem from a lack of forage or insufficient funds for the purchase of adequate feed. Pneumonia, lamb mortality due to septicemia and cold shock, enterotoxemia, and lameness are prevalent. Parasites, which sometimes cause significant losses, vary in their prevalence by season and management system. Lungworms, flukes, hydatid cysts, and strongyles are the most common parasites. Government vaccination campaigns are conducted for enterotoxemia, FMD, sheep pox, and AHS.

The general direction the Peace Corps veterinarians have taken is toward government-funded health protection programs, milk and meat inspection, and individual animal treatments. The Peace Corps believes these initiatives could lead to new animal health and husbandry extension programs in Morocco. Inquiries about the Peace Corps veterinary program may be directed to Dr. Carolyn Prouty at the address below or to Ms. Jennifer Knapp, Peace Corps, 1990 K Street, NW., Washington, DC 20526.

(Dr. Carolyn Prouty, Service d'Elevage, Bouarfa, Province de Figuig, Morocco)

#### **Hydropericardium Disease Update**

Experiments carried out during the last 2 years have suggested that hydropericardium syndrome (HpS) in chickens is caused by the interaction of two agents: an avian adenovirus and a smaller, unidentified agent. The other agent, which is less than 25 nm in diameter, caused mortality without gross hemorrhages in 7- to 9-day embryonating chicken eggs when inoculated by yolk sac, chorioallantoic sac, or chorioallantoic membrane routes. The unidentified agent did not produce cytopathic effects in chicken embryo kidney cells, chicken embryo liver cells, or QT 35 cell line.

Adenovirus from HpS was successfully grown in chicken embryo kidney cells up to seven passages. It produced round cell cytopathic effects in chicken embryo kidney and chicken embryo liver cells but did not grow in QT 35 or Vero cell lines. This adenovirus also caused mortality in embryonating eggs of broiler breeders and specific pathogen-free white leghorn chickens. Dead embryos were grossly hemorrhagic.

Development of a formalinized liver homogenate vaccine was reported in August 1988 (see 18-1: 10-11). This vaccine provided good protection in laboratory and field trials and is currently manufactured by a large number of private and public organizations. An improved vaccine for HpS was developed with higher infectious-agent contents, minimum cellular debris, and easy injectability. In an experimental challenge-protection study, this vaccine provided good protection. Mortality in vaccinated birds was zero compared to 52.9 percent in the unvaccinated controls.

[Adapted from the abstract of a paper presented by Dr. Mohammad Afzal at the Third International Veterinary Conference held in Islamabad, Pakistan, on November 29 and 30, 1990. The paper was entitled "Etiology and Control of Hydropericardium Syndrome (Angara Disease) in Broilers of Pakistan," by M. Afzal, Rakshanda Muneer, George Stein, Jr., and Barrett S. Cowen.]

For more information, readers may contact Dr. Afzal at the Animal Sciences Institute, National Agricultural Research Centre, Islamabad, Pakistan.

## **Focus on Japanese Encephalitis**

Japanese encephalitis (JE) is an arboviral encephalitis of eastern and Southeast Asia affecting swine, humans, and, to a lesser extent, horses. Swine play an important role as amplifiers of the virus in areas with large swine populations. Infected neonatal swine may die, and pregnant sows may abort. Horses may show transient CNS signs but seldom die of JE. Cattle, sheep, and goats may become inapparently infected but play no major role in JE transmission.

Clinical signs of JE in people include fever, headache, vomiting, neck rigidity, disorientation, delirium, convulsions (principally in children), paresis, and paralysis. Neurologic and psychiatric sequelae are common. Mortality rates vary between 20 and 50 percent, with death occurring in the first 10 days of onset.

### **JE Virus**

JE virus is a positive sense, single-stranded RNA virus in the family Flaviviridae, genus *Flavivirus*. Based upon crossed neutralization studies, it is believed to be part of a complex of neurotropic viruses that include those of St. Louis, Murray Valley, Rocio, and West Nile encephalitis. High titers of JE virus have been attained in various vertebrate cell lines. Strain variations have been demonstrated in monoclonal antibody and RNA genome sequence studies.

### **History**

JE was probably first described in 1871 in Japan as “summer encephalitis” in humans. The JE virus was isolated from a patient who died of encephalitis more than 60 years later. The disease was first termed “Type B” or Japanese B encephalitis to differentiate it from “Type A” or epidemic encephalitis lethargica. Seasonal occurrence indicated possible vector transmission. In 1938, Japanese investigators isolated the JE virus from wild *Culex tritaeniorhynchus* mosquitoes.

### **Geographic Distribution**

Major JE epidemics in humans have been reported throughout Asia. A 1924 outbreak in Japan resulted in 6,125 cases with 3,797 deaths. In 1949, Korea reported 5,548 cases, and in 1966, China identified more than 40,000 cases. Burke and Leake (1988) estimated that JE affects approximately 50,000 people each year in Southeast, East, and Midsouth Asia and kills about 12,500 of them.

JE has been reported from maritime Siberia, North and South Korea, Japan, China, Taiwan, the Philippines, Vietnam, Laos, Kampuchea, Thailand, Myanmar (Burma), Malaysia, Singapore, Indonesia, India, Bangladesh, Nepal, and Sri Lanka. Seasonal epidemics of JE occur primarily in temperate regions above 15° N. latitude, in China, Korea, and northern Thailand. In contrast, endemic low-level viremias and vector transmission occur continuously in tropical areas of Southeast and South Asia. Ecologically, JE occurs in an area with Murray Valley encephalitis to the east and West Nile fever to the west.

### **Transmission**

The principal arthropod vector of JE virus is *Culex tritaeniorhynchus*. This mosquito prefers feeding on domestic animals and birds but will feed on humans as substitute blood hosts. *Cx. tritaeniorhynchus* oviposits in flooded rice fields and other bodies of fresh water. It is most active at twilight hours but does not enter dwellings. Other mosquitoes from which JE virus has been isolated include *Cx. gelidus*, *Mansonia uniformis*, *Aedes curtipes*, *Culex* spp., *Mansonia* spp., and *Anopheles* spp.

Birds and swine play important roles in the spread and maintenance of JE virus. Ardeid birds, among them night herons and egrets, and certain migratory birds inhabit rural and

urban areas of high vector density. The virus readily circulates in and sometimes spreads from these endemic areas.

Swine are key amplifiers of JE virus in many parts of Asia. They provide a large vector-accessible population and fast turnover of generations, thereby assuring a continuous supply of susceptible animals that commonly develop high-titer JE viremias of 2 to 4 days' duration. Viral transmission to gilts also has been demonstrated in sperm from experimentally infected boars.

Various domestic and wild animals have been shown to have JE virus antibody but are not considered significant factors in virus propagation. Horses and cattle develop low-level JE viremias. Serological surveys have shown extensive infection in these animal populations. Epidemiologic studies in Japan did not support the hypothesis that snakes carry JE virus, but subsequent work in Korea demonstrated overwintering virus in snakes, frogs, and bats. Several attempts have been made to show that bats can serve as JE virus hosts. Even though bats have produced viremias capable of infecting mosquitoes, there is no clear evidence that bats propagate JE.

### Clinical Signs

The most significant agroeconomic losses caused by JE are from abortions and neonatal mortality in swine. Adult swine may experience low-grade fever and loss of appetite. Neonatal mortality rates of 50 to 70 percent were reported in Japan during a 1947–49 epidemic. Fetuses are aborted at varying stages of development. Some may be mummified. Newborn piglets may be weak and unable to stand and may have tremors and convulsions. Seemingly normal newborn animals may later demonstrate CNS abnormalities before they die. At necropsy, salient gross lesions are neurologic and may include spinal hypomyelinogenesis, meningeal congestion, encephalomalacia, hydrocephalus with thinning of the cerebral cortex, and hypoplasia of the cerebellum.

Horses affected with JE are often asymptomatic. Initial clinical signs are similar to those seen in the equine encephalitides; however, the mortality rate is generally less than 5 percent. The incubation period is from 8 to 10 days with subsequent signs lasting 4 to 9 days. Fever, photophobia, bruxism, incoordination, irregular gait, ataxia, and stupor are among the initial signs of the disease. Severely affected horses may develop blindness and coma and ultimately die.

No abnormal signs have been described in cattle, sheep, or goats affected with JE, unlike Borna disease.

Studies of JE antibody levels in Asia indicate that a majority of cases in people are subclinical without neurologic involvement. When the disease reaches clinical levels in humans, symptoms resemble those of acute meningomyeloencephalitis. An incubation period of 1 to 2 weeks usually precedes cephalgia, hyperpyrexia, stupor, and, in affected children, motor seizures. The examination of cerebrospinal fluid reveals a lymphocytic pleocytosis with concurrent elevation of pressure and total protein.

A partial list of diseases that may resemble JE in swine includes pseudorabies, porcine parvovirus disease, hog cholera, Teschen disease, Talfan disease, porcine hemagglutinating encephalomyelitis, and porcine polioencephalomyelitis. In Japan, a hemagglutinating DNA virus has been isolated from swine with a disease process

that resembles JE. The list of equine diseases that resemble JE should include eastern, western, and Venezuelan encephalitis in the Americas, Borna disease, Maingrain virus disease, rabies, protozoal myeloencephalitis, verminous encephalitis, and toxic encephalitides.

## Diagnosis

Definitive diagnosis of JE is based on viral isolation or detection of viral antigen by enzyme-linked immunosorbent assay (ELISA) or immunofluorescence techniques. Preferred specimens for virus isolation are (1) brains from piglets that have been aborted, are stillborn, or died after demonstrating signs of encephalitis; and (2) placenta.

Specimens intended for attempted viral isolation should be packed in wet or dry ice and sent to a laboratory with arboviral diagnostic capabilities. A separate set of specimens should be fixed in buffered formalin and sent for histopathological examination.

Serum samples (10 ml) should be collected from stillborn or unsuckled piglets showing disease signs.

Paired acute and convalescent serum samples can be used to demonstrate JE seroconversion by hemagglutination inhibition, virus neutralization, and complement fixation.

Seroconversion is difficult to demonstrate in dams; however, detectable levels of macroglobulin (IgM) antibodies are present in JE virus-infected sows after they have farrowed abnormal litters.

An IgM capture ELISA has been developed to detect early JE virus infection in pigs and horses. The JE IgM ELISA has nearly replaced hemagglutination inhibition as a human diagnostic test and has been adapted as a field kit. The ELISA technique is the principal serological test for JE in horses.

Macroscopically and microscopically, equine cases of JE are indistinguishable from American and Venezuelan encephalitides of horses. Microscopic changes include inflammatory changes of the brain stem, cerebral cortex, white substance and basal ganglia, and interstitial infiltration with varying degrees of dispersed neuronal degeneration and perivascular cuffing. Throughout the gray and white matter, round cells associated with microglia form focal glial or neuronophagic nodules. Cerebellar inflammatory changes are restricted principally to the Purkinje and molecular strata.

## Prevention and Control

Vaccines are used to prevent JE in swine, horses, and humans. Vaccination has been credited with drastically reducing JE throughout Asia. The use of vaccines in swine carries a twofold benefit by preventing both reproductive losses and viral amplification. The recommended schedule for attenuated vaccine in swine is two inoculations for young gilts and boars and one inoculation for sows before the peak mosquito season. Both inactivated and attenuated virus vaccines are available for horses. Over a half million horses were given attenuated JE virus vaccine in China, reportedly with good protection.

Studies have shown that two doses of inactivated vaccine will afford 95-percent protection of humans with no apparent complications. Chinese researchers have also conducted safety and efficacy tests in humans on a live-attenuated JE virus vaccine with promising results.

An understanding of JE epidemiology is required in order to develop effective strategies to interrupt viral transmission. In attempts to reduce abortions and neonatal mortality in pigs, modifying some husbandry practices has proven effective. Changes include restricting service of gilts to periods of low vector density and placing mosquito netting over pens where pregnant swine are kept. Stabling at dawn and dusk, especially during seasons of peak mosquito activity, can reduce the frequency of JE transmission to horses.

## Research and Development

The World Health Organization has reported successful JE vector control by means of ultra-low-volume aerial spraying.

The ideal JE vaccine would be safe, inexpensive, and capable of inducing long-lasting protection following a single oral dose. Recent advances in recombinant DNA research should facilitate development of such a vaccine. These second-generation vaccines will probably be available for pigs and horses before the vaccines can be approved for use in humans. Current approaches involve cloning of genes coding for the protective E and NS-1 glycoproteins of JE virus and incorporating those genes into a live vector, such as *Vaccinia* virus, or into a chimeric system, such as hepatitis B core particles. It may also be possible to develop a full-length infectious clone of JE cDNA that would permit site-directed changes in the genome leading to specific attenuating lesions. Oral immunization of pigs would avoid fetal losses due to JE, eliminate a major source of virus amplification, and diminish epidemic spread to horses and humans.

Similar recombinant DNA technology is being used to engineer specific diagnostic antigens and to study genetic differences in JE strains from different geographic sites. Both oligonucleotide fingerprint and limited primer extension sequencing of the RNA genome show differences between strains from the temperate epidemic regions and from the tropical endemic regions. These differences support the hypothesis that JE virus overwinters in the temperate zones and is not reintroduced each year from the Tropics.

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